AN OBSERVATION ON THE JUGULAR PULSE OF MAN.[1]

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The revival of interest in the venous pulse of man, following the publication of James Mackenzie's[2] work, has, as a natural result, given rise to considerable discussion as to the cause and significance of the various waves which go to make up the venous pulse. Controversy arose over the first or auricular wave of the jugular pulse, the *a* wave of Mackenzie, while the origin and time of the third large wave, Mackenzie's *b*, occasioned even greater dispute. The second wave of the jugular pulse, wave *c*, which interrupts the first negative phase after the auricular wave, was formerly ascribed to numerous causes; after Mackenzie's publication, however, these seemed disposed of and his explanation, namely, that wave *c* was due to the impact of the neighboring carotid, was generally accepted. As recently as the early part of 1906, Wenckebach,[3] in an exhaustive article on the venous pulse, stated that he agreed with Mackenzie and all other recent writers in regarding the wave called *c* as chiefly a carotid product. Today such unanimity no longer obtains, since within the last year Leon Fredericq[4] and Morrow[5] have sought to disprove the carotid origin of the wave *c* and, more recently, Bard,[6] Cushny and Grosh,[7] and Hirschfelder[8] have expressed a like doubt.

Briefly stated, the arguments in favor of the carotid origin of wave *c*, as advanced by Mackenzie,[9] Wenckebach, and others, are: (1) Its time falls within the time of active ventricular systole, synchronous with the carotid pulse. (2) The size of this wave varies significantly according to the nearness of the pressure point of the receiving cup to the carotid artery, and according to the depth at which it is placed in the neck tissue. (3) The wave *c* is absent in the "liver pulse." (4) During irregular heart action, when the venous pulse continues and the arterial pulse is absent, in tracings of the jugular pulse taken at this time the carotid wave is not seen. (5) When there is a distended jugular bulb the *c* wave is not apparent in the venous tracing. (6) When the carotid wave is very prominent in a jugular tracing, its shape often is almost identical with that of a tracing of the carotid pulse. Although some of these observations have been challenged, as yet no adequate proof of their inaccuracy has been advanced.

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[1] Read by invitation at a meeting of the Section on General Medicine of the College of Physicians of Philadelphia, November 11, 1907.

[2] The Study of the Pulse. Edinburgh and London, 1902.

[3] Beiträge zur Kenntnis der menschlichen Hertztätigkeit. Archiv f. Anatomie und Physiologie, 1906, Physiol. Abtlg., p. 297.

[4] La seconde ondulation positive (première ondulation systolique) du pouls veinieux physiologique chez le chien. Bulletin de l'Académie Royale de Médicine de Belgique., 1907.

[5] The Various Forms of the Negative or Physiological Venous Pulse. Brit. Med. Jour., December 22, 1906, p. 1087.

[6] Des divers details du pouls veineux des jugulaires chez l'homme. Journal de physiologie et de pathologie générale. Paris 1906, viii, 466.

[7] The Venous Pulse. Jour. Amer. Med. Assoc., October, 12, 1907.

[8] Some Variations in the Form of the Venous Pulse. Johns Hopkins Hospital Bulletin, June, July, 1907.

[9] The Interpretation of the Pulsations in the Jugular Veins. Amer. Jour. Med. Sci., 1907, cxxxiv, 12.

It is a noteworthy fact that those observers who most vigorously oppose the accepted cause of wave *c*, almost without exception, have based their observations on experiments performed upon animals in which the jugular veins were dissected out and isolated. They lose sight of the fact that the *c* wave under discussion is one obtained from the surface of the neck of man, where the veins make their changes in volume felt in the overlying structures. It is evident, therefore, that comparison is being made between phenomena which are not identical.

Morrow took tracings from the exposed right jugular of a dog in which the right carotid had been clamped below the level from which the tracing was taken. The left carotid was taken simultaneously, but no cardiogram was made with which to compare the jugular tracings. In these tracings Morrow obtained a small wave following the auricular one and occurring at the time of the beginning of the carotid pulse. He assumed that this small wave was *c* and that, all carotid influence having been eliminated, it must be caused by some other factor. He advanced several theories as to its cardiac origin.

By far the most complete experimental work has been done by Leon Fredericq. On anesthetized dogs Fredericq exposed the carotid and external jugular, and, after the vein was protected from all transmitted pulsations, he recorded simultaneously tracings of the venous pulse and of the carotids and a cardiogram of the right heart. Immediately following the first or auricular wave he observed a small second rise, which he called *bc*, which in its time corresponded a little late with the first part of the up-stroke of the cardiogram, and more or less with the beginning of the carotid pulse.

Fredericq next demonstrated that *bc* occurred in tracings of the right auricle and superior vena cava near the auricle, and, further, that at this level the beginning of *bc* coincided with the beginning of the systole of the right ventricle, but preceded the beginning of the carotid pulse. From these facts he concluded, that, although *bc* is produced earlier at the heart than the arterial pulse, since the arterial pulse, *i. e.* the carotid pulse, travels 6 to 8 meters per second whereas the venous pulse travels only 2 meters per second, the arterial pulse overtakes the wave *bc* at the level of the neck. Hence the coincidence in time which is usually observed, a fact which Fredericq infers accounts for the seeming relation which exists between the *c* wave in the jugular pulse of man and the carotid pulse. The other writers who doubt the carotid origin of *c* apparently base their doubts largely on the above-mentioned experimental work.

It is obvious that both Morrow and Fredericq effectually eliminated all possibility of the carotid influencing their jugular tracings, and they certainly demonstrated that in the neck veins of dogs, as Chauveau has shown for the horse, there is a wave following *a* which coincides with the beginning of ventricular systole. It seems, however, by no means proved that the usually prominent *c* wave of the jugular pulse of man, occurring at a time when ventricular systole is fully established, is identical with this small wave which occurs earlier in its relation to the cardiac cycle.

It seemed, therefore, important to determine, if possible, the presence or absence of a wave in the jugular pulse of man which should correspond in the time of its occurrence to the wave *bc* of Leon Fredericq; and thus, if found present, to demonstrate that this wave, which Fredericq and Morrow confused with the *c* wave, exists in man as well as in dogs and occurs along with and entirely independent of the carotid wave.

On examining a clear cardiogram, there will often be found a little notch on the up-stroke of the tracing. The interval between the exact beginning of the rise due to ventricular systole and the top of this notch represents that part of the ventricular systole during which the ventricular pressure is steadily being raised until the hitherto closed semilunar valves are opened and active systole begins. The notch marks the opening of the semilunar valves, and the interval before this occurs may be termed a *ventricular presphygmic period*, or, as it has been called by some, the "*Anspannungzeit.*" It will be noticed that the wave *bc* of Fredericq occurs during this period of the cardiogram.

Therefore, in seeking a like wave in man only those tracings were used in which, if not a clear ventricular presphygmic period (Anspannungzeit) occurred, at least a suggestion of one was present in the cardiogram, and in which the venous pulse was clear. Some difficulty was experienced, due to the fact that the exact beginning of ventricular systole is often difficult to determine accurately. Further, it must be borne in mind that as the ventricular presphygmic period is an event recorded at the heart itself, any event in the jugular pulse which corresponds to this period in the cardiac cycle must be represented on the tracings as occurring slightly later. Such retardation was observed by Professor Wenckebach and me, but our estimate of the time of the delay was less than that recently given by Hewlett.[10]

The tracings examined were taken from persons normal as to their cardiovascular systems, as well as from those with grave lesions of the heart or kidneys. A number of jugular tracings showed a second distinct wave following the *a* wave and preceding the *c* wave (Fig. 1). But, upon carefully measuring the exact time of this wave, it was found that often it coincided exactly with or actually preceded the ventricular presphygmic period.

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[10] The Interpretation of the Positive Venous Pulse. Jour. Med. Research, October, 1907.

Fredericq has shown that his wave *bc* was originated by the beginning of ventricular systole. He demonstrated that it was present when the auricles ceased contracting and the ventricles continued to beat; and that when ventricular contractions ceased the wave also disappeared in spite of auricular systole going on. It is evident, therefore, that those waves which in the tracings studied by me fell with or before the ventricular presphygmic period, could not be the same as Fredericq's wave, since, in the jugular pulse, at the period in question, that is before the ventricular presphygmic period, the influence of ventricular systole is not yet evident.

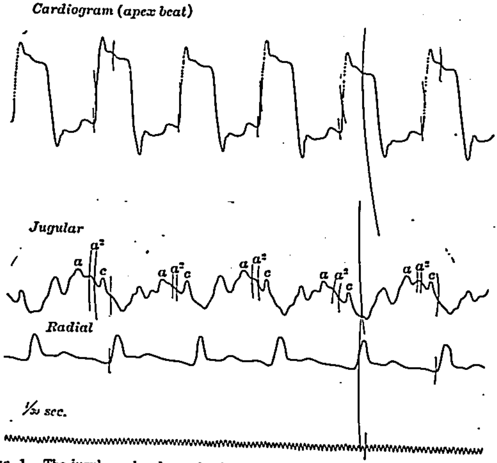

FIG. 1.—The jugular pulse shows the doubling of the auricular wave. The second rise of this (a^2) falls in or before the ventricular presphygmic period, which is indicated by the markings on the cardiogram.

That a wave had to be dealt with which was in the nature of a double *a* wave seemed all the more certain, since in some cases of heart block a beautifully clear wave was found, which followed immediately after the waves due to auricular systole, even when the latter were not succeeded by any ventricular contraction. Consequently, the influence of any ventricular systole as a factor in the cause of these double *a* waves was absolutely excluded in such cases. An illustration of this is seen in Fig. 2. It was evident, therefore, that care had to be taken not to confound a double *a* wave with the wave which was being sought.

The examination of other tracings revealed, in addition to this doubling of the *a* wave, still another small wave which fell just after or in the latter part of the ventricular presphygmic period, but invariably came before the beginning of active ventricular systole and hence preceded the *c* wave. Such a tracing is shown in Fig. 3, in which, at the bottom of a double auricular wave, a small but distinct wave is seen, designated by the letter *b*. By using the radial pulse as a means of comparing events in the jugular pulse and cardiogram, it is found that *b* occurs just after the ventricular presphygmic period. The foregoing seemed the proof that, aside from any double *a*, there exists in jugular tracings of man a small wave which corresponds in time to the wave found by Morrow and Fredericq in dogs, and which is clearly separate and distinct from the carotid wave (Mackenzie's *c*).

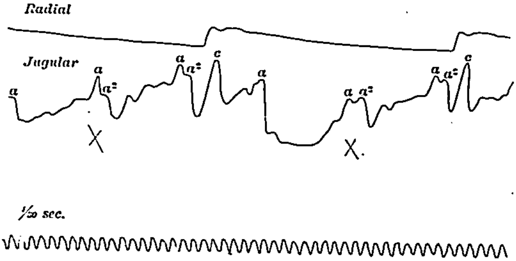

Fig. 2.—Heart block in which the jugular pulse shows a double *a* wave (a^2), even when the auricular contraction is not succeeded by a ventricular systole, as at (X).

The occasional presence of the wave in question has been noted in a recent paper by L. Bard. In at least one of his tracings, he points out a wave in regard to the identity of which, with the one under consideration, there is little doubt. Bard called this wave *i*, but in view of the newer lettering of the venous pulse adopted by D. Gerhardt, Wenckebach, and others, in which one of the postsphygmic waves is called *i*, it was suggested by Prof. Wenckebach that this small second wave of the jugular pulse of man might appropriately be called wave *b*. We should have, therefore, the following convenient sequence of lettering: *a*, the auricular wave; *b*, the wave above described; *c*, the carotid wave; and *d*, the

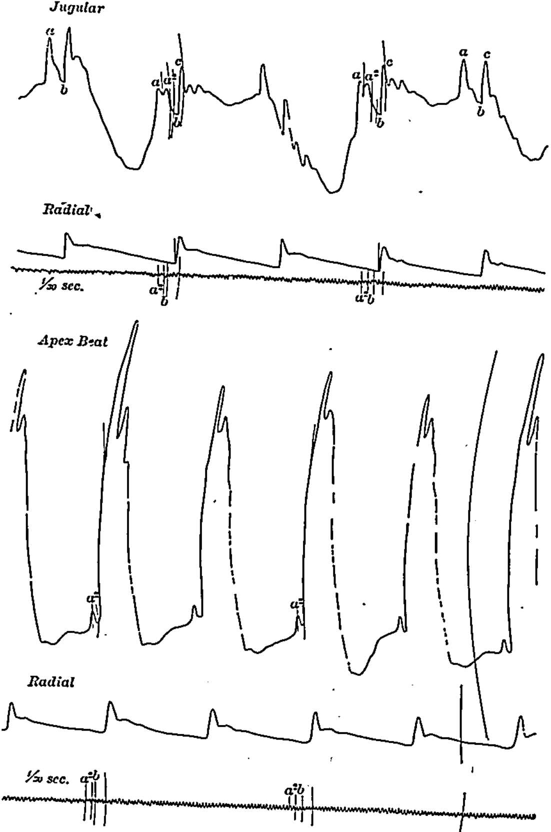

FIG. 3.—In the jugular pulse there occurs at the bottom of the double *a* wave another small wave, *b*. Wave *b* occurs just at the end of the ventricular presphygmic period and always precedes the beginning of *c*. The jugular pulse and the apex beat must be compared by means of the radial pulse.

diastolic wave of Wenckebach and Gerhart,[11] erroneously called v. The letter v should be reserved for such phenomena as occur during ventricular systole only. In a paper which appeared about

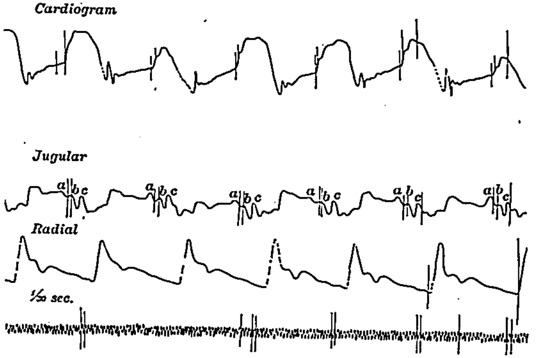

FIG. 4.—Wave b occurs as a distinct wave interrupting the down stroke of the a wave. It always occurs just after or in the last part of the ventricular presphygmic period, which is clearly shown on the cardiogram.

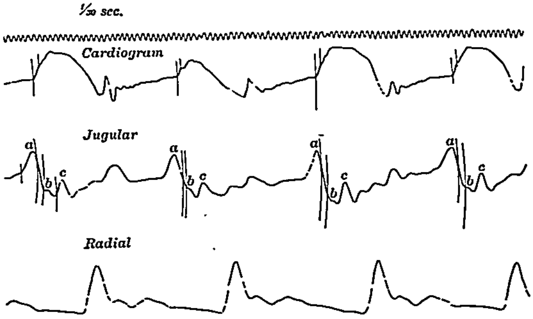

FIG. 5.—Wave b occurs just after the ventricular presphygmic period and is seen as a distinct elevation between the bottom of the down stroke of the a wave and the beginning of wave c.

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[11] Klinische Untersuchungen über Venenpulsationen: Archiv. f. exp. Pathologie u. Pharmakologie, 1894, xxxiv, 402.

the same time as Bard's, Hirschfelder[12] makes no mention of the *c* wave as such, but describes, as the second wave of the jugular pulse, a wave similar in time to the above noted *b* wave; none of the tracings in this paper, however, seem to show a *b* wave.

The double *a* being of comparatively rare occurrence, the additional wave *b* was found most often following the usual single auricular wave. Its character was by no means constant, as may be seen from the accompanying series of tracings. In Fig. 4, *b* appears as a distinct wave interrupting the down stroke of *a*. In Figs. 5 and 6 an obvious wave is seen between the bottom of *a* and the beginning of *c*. Not infrequently *b* shows more as an outbowing on the descending limb of *a* than as an abruptly rising wave. Of this type Fig. 7 furnishes an example. Again, there are tracings which show no clear *b* wave, but in which, during a distinct interval before the beginning of *c*, there is the suggestion of a small wave *b* (Fig. 8). Finally, *b* may appear as a wave at the beginning of the up-stroke of *c*. Fig. 9 shows this, although in this tracing the ventricular presphygmic period is not clear; nevertheless, *b* obviously falls in the first part of the systolic ventricular rise of the cardiogram. In spite of these variations as to form, the time of *b* remains unchanged. In

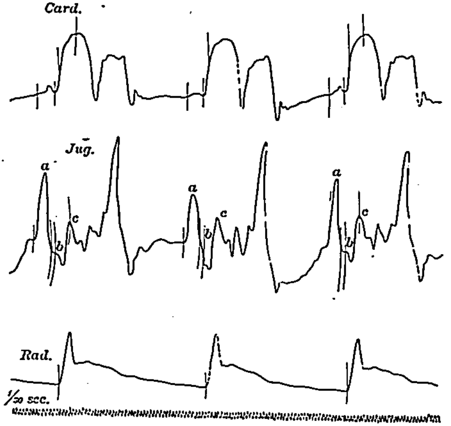

FIG. 6.—Wave *b* occurs at the bottom of the *a* wave when the latter follows a pause after an extra systole.

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[12] Graphic Methods in the Study of Cardiac Disease. AMER. JOUR. MED. SCI., 1906, cxxxii, 378.

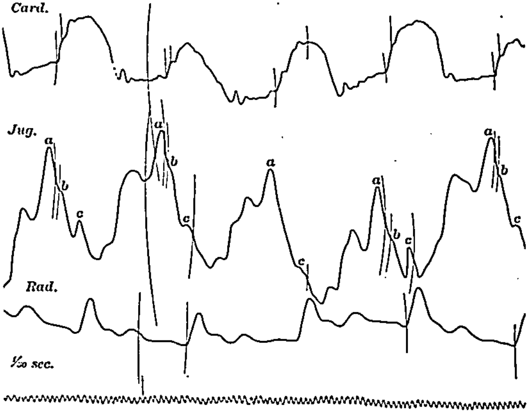

FIG. 7.—Wave *b* appears on the down stroke of the *a* wave more as an outbowing than as an abruptly rising wave, but its relationship to the ventricular presphygmic period is always correct.

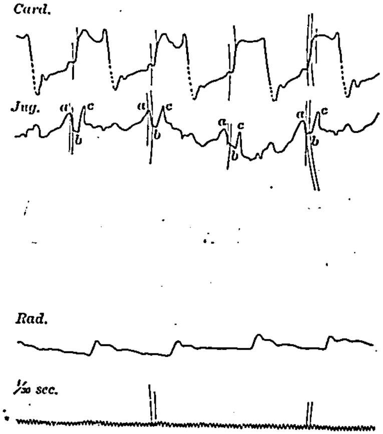

FIG. 8.—There is no distinct *b* wave in the jugular pulse, but in the interval between the end of *a* and the beginning of *c* the suggestion of one is evident.

the tracings examined it always occurred just after or in the last part of the ventricular presphygmic period.

This wave *b* is always small and by no means apparent in all jugular tracings, but when present can be no mere accident, since it is observed not only throughout a single tracing, but in repeated tracings from the same individual, even when they are taken on different occasions. Its occurrence is probably somewhat dependent upon the length of the *a-c* interval and the prominence of the *c* wave.

The cause of wave *b*, in the absence of definite experimental proof, remains a matter of speculation, although there seems no good reason for not accepting the suggestion of Leon Fredericq, namely, that this wave is due to the augmentation of the intraventricular pressure before the opening of the semilunar valves, and that this increased pressure communicates itself to the contents of the auricle across the auriculo-ventricular orifice closed at this moment by the tricuspid valves whose leafletts bulge toward the right auricle. Bard gave a like explanation of the cause of his wave *i*, and Hirschfelder attributed the second wave of the venous pulse to these same factors.

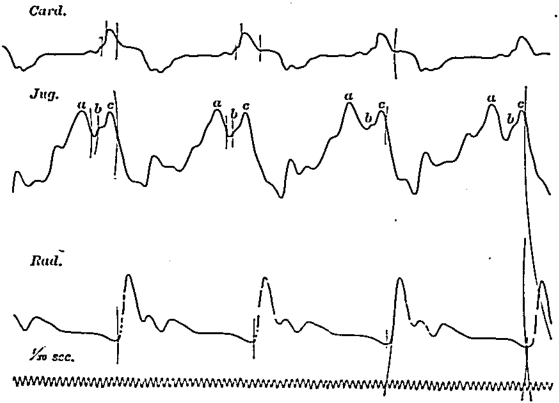

FIG. 9.—Wave *b* appears as a distinct elevation on the first part of the ascending limb of wave *c*. Although the ventricular presphygmic period is not clear in the tracing, wave *b* obviously falls in the early part of the systolic ventricular rise of the cardiogram.

From the foregoing considerations the following conclusions seem warranted:

1. There are not sufficient grounds to doubt that the *c* wave in the jugular pulse of man is, as Mackenzie said, anything but a carotid product.

2. The jugular pulse of dogs, even when all carotid influence is eliminated, shows a small wave at the beginning of ventricular systole.
3. In man a double *a* wave is sometimes found, the second rise of which cannot be identical with the wave described in dogs, since its occurrence is too early.
4. In addition to any double *a* wave in man, another wave, appropriately called wave *b*, often shows between *a* and *c* and begins just after or in the last part of the ventricular presphygmic period.
5. Since the wave found in the jugular pulse of dogs at the beginning of ventricular systole has its counterpart in the *b* wave of man's jugular pulse, the assumption that the wave found in dogs is identical with Mackenzie's *c* wave is unjustifiable.

I gratefully acknowledge the many kindnesses shown me by Prof. K. F. Wenckebach, at whose suggestion this work was undertaken. In addition to the admirable facilities afforded in the Laboratory of Clinical Medicine at Groningen, Prof. Wenckebach generously placed at my disposal for study a large series of tracings, which, together with those that I made myself, supplied the material for the observations above recorded.

PRIMARY CARCINOMA OF THE VERMIFORM APPENDIX.

A STUDY OF 90 CASES, 3 PREVIOUSLY UNPUBLISHED.[1]

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The increasing reports of isolated cases of primary carcinoma of the appendix, which one finds in the literature, testify not only to the greater interest now manifested in the study of the pathological anatomy of that organ, but also to the greater care exercised in its examination, whether removed at operation or on the autopsy table. A sufficient number of such neoplasms have now been recorded to make a critical study of them interesting and instructive. To the 87 cases that I have found reported in the literature, I desire to add the following 3 cases:

Case XXII.—An unmarried, German, immigrant girl, aged twenty years, was admitted, on September 5, 1907, directly from the steamer to the Presbyterian Hospital, to the service of Dr. F. T. Brown, to whom I am indebted for the privilege of taking care of

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[1] The reports of 15 additional cases were found after this article had been completed. Since it was impossible to include them in the detailed study, they have been added in an appendix. The total number of cases reported, therefore, is 105.